TABLE 1.—*Major Immediate Causes of Climbing Accidents*

	Percent
Fall or slip on rock	29
Slip on snow or ice	20
Avalanche	11
Pulmonary edema, frostbite, other illness	8
Fall into crevasse	8
Hypothermia	2.5
Contributory causes were:	
Exceeding ability	29
Climbing unroped	22
Inadequate equipment	7
Party separated	3

the lower 48 states as well, particularly in the Rockies.[2] The incidence in persons less than 20 years old is approximately 9 percent but it is lower in adults, being approximately 0.6 percent.[3,4] Deaths due to hypothermia and exposure are occurring more frequently in the western states due to an increasing number of winter climbs and cross-country ski trips.[5,6]

The variety of accidents that can occur in climbing is well documented in Wilson's paper. In 1976 the American Alpine Club received reports of a total of 405 persons involved in 214 accidents in the United States.[7] There were 51 deaths.

The major immediate causes of the accidents are shown in Table 1.

In all, 134 persons (63 percent) were aged 15 to 30 years and 19 percent were between 15 and 20 years old.

This is not a complete survey of all climbing accidents occurring in the United States because only a small proportion of accidents are reported to the American Alpine Club.

Despite these gloomy statistics one can enjoy mountaineering safely and age is not a limiting factor. Ricardo Cassin at 66 years of age was still climbing with big Himalayan expeditions. Richard Hechtel of Redwood City, California, at 64 years of age is still climbing big mountains and in 1975 climbed Illimani in Bolivia (21,201 feet). John Graham of Santa Barbara ascended Mt. Denali (20,320 feet) at the age of 65.

A few general suggestions regarding safety in climbing are appropriate:

- Climb for enjoyment. Do not climb to be a hero.
- Know when to turn back.
- Travel with a guide or an experienced (preferably older) climber. Don't climb alone.
- Climb with good equipment and know how to use it.
- Take a climbing course in theory, methods and practical experience. The Sierra Club, The Seattle Mountaineers and the American Alpine Club hold annual courses on various aspects of mountaineering. Practical climbing courses are offered widely in the western states.

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Modern View of Ataxia

ATAXIA IS A SIGN commonly found as a result of the acute and chronic toxicity of ethanol and of such anticonvulsants as phenytoin (Dilantin®). Even if these cases are not counted, ataxia is a frequent presenting sign of a number of acquired and inherited diseases. Though the inherited diseases as a group occur frequently, many of the individual inherited diseases that give rise to ataxia are rare and are well known only to a few specialists. The first descriptions of the inherited ataxias were recorded more than a century ago and they once helped to make neurology a specialty distinct from internal medicine and from psychiatry. This field, the inherited ataxias and the acquired diseases which mimic them, has only recently matured to an independent status in modern neurology. The underlying disorders have recently been classified, loosely, as defects in oxidative metabolism, in lipid catabolism, in the metabolism of small molecules containing nitro-

gen, and as slow infections from atypical viruses.[1] Few current textbooks discuss the diseases or their biochemical and viral causes clearly and it is difficult to find an adequate list of the acquired diseases that need to be considered in any differential diagnosis.

For these reasons, the major contributions of the Specialty Conference elsewhere in this issue on cerebellar ataxias are a clear, concise synopsis of modern knowledge of the ataxias and a modern differential diagnosis of the acquired and inherited disorders of coordination and balance. While Dr. Dreyfus and his colleagues correctly call their classification arbitrary, any classification today must be arbitrary, given our limited knowledge of the causes and the pathogenetic mechanisms of these diseases. Even with dissension on details, the classification given by the conference is useful in calling attention to the areas of diagnosis and the breadth of underlying problems that must be considered in ataxia patients. In addition, the conference includes brief, but lucid, accounts of the complexities of cerebellar anatomy and physiology, which emphasize the essential points needed to understand the clinical abnormalities.

A few minor points will cause disagreement. The sporadic incidence of late-onset cortical cerebellar degeneration, and of many other "inherited" ataxias, is consistent with incomplete penetrance of a dominant mutation, as suggested in the conference, but it is equally consistent with a recessive mutation or one or more exogenous causes of disease. Most authors[1,2] define Friedreich's ataxia more narrowly than Dr. Vijayan does. Usually the age of onset is limited to a few years before or after puberty. The disease is described as having a recessive or sporadic pattern with ataxia of the legs progressing to the arms, to speech and to the eyes in that order over a decade or more, with impairment of the senses of position and vibration and loss of the deep-tendon reflexes from the time the patient is first examined, with subsequent loss of muscle bulk and strength (in either a distal or a proximal pattern), and with subsequent reduction in the senses of light touch and pin-prick. As mentioned in the Specialty Conference, pes cavus and kyphoscoliosis are almost always found and there is evidence of cardiomyopathy at autopsy, if not before. In fact, the disease is strictly defined pathologically, not clinically.[1,2] The syndrome is associated with dementia even in its early stages.[1-3] Finally, several authors have been more impressed with the signs of mental retardation, dementia or failure of intellectual maturation in ataxia telangiectasia[4,5] than in a-beta-lipoproteinemia,[6] but it is difficult to test mental function adequately in a patient with slow, ataxic movements and speech.

No doubt these differences of opinion will be clarified by further research. Meanwhile, it is important for the patient's sake that physicians recognize the ataxias; know that modern techniques such as radiology, genetic linkage studies and biochemistry can often aid in diagnosis or prognosis; be able to treat according to the precise diagnosis, and be able to refer patients, when appropriate, to centers where clinical research and special forms of treatment may be available.

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